The Karlsruhe Heart Model KaHMo: A modular framework for numerical simulation of cardiac hemodynamics

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***Abstract*—Numerical methods are rapidly gaining importance for answering medical questions. One field in which these answer are especially valuable is cardiology. The understanding of the cardiac function on a detailed, physical level can help to improve diagnostics, prognosis and therapy for a large number of pathologies.**

The KaHMo (Karlsruhe Heart Model) is developed as a framework for the patient specific numerical simulation of the intraventricular flow. The framework combines different methods from several disciplines.

As a means to simulate the cardiac flow in a given patient specific heart, KaHMo MRI derives the time dependent geometry of the endocardium and performs a numerical simulation of the intraventricular flow.

In order to be able to predict the influence of pathological changes in e.g. the myocardium or the valves on the contraction of the heart and the flow driven by this movement, KaHMo FSI employs special Fluid-Structure-Interaction methods and a composite approach to muscular dynamics to simulate the complex interaction of non linear elastomechanics with hemodynamics.

The framework is supported by additional models which include a model of the human circulatory system to derive the systemic pressure response, rheological models for the non-Newtonian behaviour of the blood as well as models for prediction of hemolysis and thrombosis risks in artificial blood pumps or ventricular assist devices.

Future developments may incorporate electrodynamical models to include the possibility to predict the effect of e.g. arrythmia or therapeutical ablation on the heart function.

The vision is a macroscopic holistic model of the human heart that can help to answer the ever pressing "what if?" questions.

Keywords—**patient specific, heart model, computational fluid dynamics, fluid structure interaction.**

I. Introduction

Heart diseases are statistically the major cause of death in the western world. To improve diagnosis and therapy methods in the field of surgical heart failure treatment, the Institute for Fluid Mechanics, University of Karlsruhe, in cooperation with the Department of Cardio-vascular Surgery, University of Freiburg, has been developing KaHMo (Karlsruhe Heart Model), a patient-specific numerical model of the human heart [1-5].

From a fluid dynamicists point of view the numerical models of the heart can roughly be divided into two types:

Firstly those that take the movement of the inner ventricular wall as a boundary condition: The prescribed geometry models [6-12].

Secondly, the models which take into account the interaction between the fluid flow and the motion driving it: The Fluid-Structure-Interaction (FSI) models, including fictitious domain models [13-16] and coupled FSI models [17-22].

Both models cannot be considered as alternatives to each other but as approaches in their own right.

KaHMo strives to combine the features of both approaches by modularization. Fig. 1 shows the principle concept map of KaHMo.

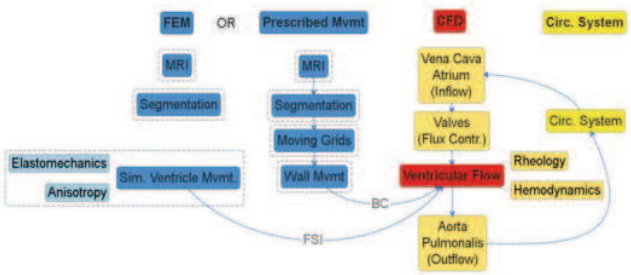

Fig. 1 KaHMo – concept map of heart simulation for the prescribed geometry (MRT, right) and fluid structure interaction (FSI, left) approach

For KaHMo MRT, the time dependent geometry of the ventricular inner walls is based on the segmentation of MRI recording sets. This information is used to generate surface meshes of identical topology to prescribe the ventricular geometry motion as moving wall boundary conditions in a Computational Fluid Dynamics (CFD) domain. For KaHMo FSI, the same fluid domain is used, but instead of the moving prescribed boundary condition a full solid mechanics model of the heart muscle is created and coupled to the CFD domain.

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This shows the modularity of the concept which enables us to create both types of models without any changes to the fluid model itself.

II. Materials and methods

Flow simulations are performed using the commercial finite volume package StarCD® (cd-adapco). In future work FLUENT® (ANSYS) will be used, which provides special capabilities to handle large mesh deformation using powerful smoothing and remeshing algorithms on unstructured meshes in order to provide better mesh quality and avoid the early termination of the fluid solver due to large cell distortion, which is a general problem in biofluid mechanics CFD simulations [23].

While in StarCD® structured volume meshes are used, MpCCI® (Mesh based Code Coupling Interface by Fraunhofer Institute of Scientific Computing and Algorithms) is used to couple the vertex motion of structured surface meshes to an unstructured volume mesh in FLUENT.

For the MRT model a special MpCCI Code Adapter was developed [24]. For the FSI model a special implicit coupling procedure is used which has been developed since the explicit methods available in MpCCI® are not stable for the given ratio of fluid and tissue densities due to the added mass effect [25,26].

A. Numerical Model

The simulation of the fluid flow is performed using the finite volume method in the arbitrary Lagrange-Euler (ALE) formulation for moving grids.

Though blood is a non-Newtonian fluid the viscous stress tensor is still being modeled by the Stokes Assumption with an effective viscosity modeled by a Carreau or Cross model adapted to measurements by *Liepsch* [27,28].

Relative pressure boundary conditions are used at the inlet to the atrium and at the exit of the aortic tract. The pressures are calculated from the volume flux at the aorta, which is used as input to a full model of the circulatory system which gives realistic pressures over time for the inlet and outlet boundaries [29,30].

The valves are represented by a two dimensional planar model. Numerically the valves are defined by a temporally and spatially variable pressure drop. The pressure drop is varied from infinite (closed valve area) to zero (open valve area). An intermediate pressure drop is defined for the border of the opening area, to smoothen the resulting jet profile in the same way as the three dimensional valve geometry does.

An exhaustive description of the flow model can be found in [2].

For the prescribed geometry model the time dependent geometry is derived from the MRI scans of a healthy male volunteer. 17 structured surface meshes were generated equally distributed over one cardiac cycle. Surface motion between these 17 base meshes is approximated by third order segmented Bezier curves. The segments are steady in terms of parametric differentials, which ensures that no unrealistic pressure peaks occur due to sudden surface accelerations in the flow solution [2,30].

For the FSI model a composite model of the myocardium was used. In this model the anisotropic behavior of the muscle tissue was implemented by inner and outer fiber layers on an isotropic matrix [31-33]. The contraction of the tissue was modeled by reduction of the fibre length corresponding to the shorting of sarcomeres.

III. Results and discussion

Figure 2 shows the simulated flow field for the FSI and MRT (StarCD®) models at the end of the diastole. In the middle pane the three dimensional momentary streamlines are presented. In the left pane two dimensional streamlines in the central plane and in the left panel the isosurface of $\lambda_2 = -1000$ are shown. The streamlines and isosurfaces are colored by the velocity magnitude.

At the end of the diastole we see a clockwise vortex that fills nearly all room in the ventrice but the apex. In the apex a second vortex develops which is responsible for flushing old blood out of the tip of the ventricle.

Figure 3 shows a schematic of the simulated flow fields for the 2 models. In the beginning diastole blood enters the ventricle through the opening mitral valve. The resulting jet flow develops into a ring vortex. This initial vortex is born from the jet shear layer that is rolled up by viscous forces exerted from the resting fluid onto the jet core. This process is well known from accelerated flow through an anular orifice. Initially this ring vortex is nearly symmetrical and corresponds to the shape of the valvular opening. In a symmetrical vessel the ring vortex would keep travelling downstream in its symmetrical shape and finally dissipate - possibly deformed by instability waves.

In our case the ring vortex originates from a laterally displaced orifice and enters an asymmetric ventricle. This leads to asymmetric growth of the ring vortex in the course of the diastole and finally the vortex is tilted to fill the elongated shape of the ventricle. The direction of the tilting is subject to boundary conditions, especially the inflow direction and the movement of the ventricle wall. In the left ventricle the tilting direction is clockwise such that the left side of the

ring vortex stays in the upper part of the ventricle and grows to fill most of its cavity and the right side dives into the apex and aids in flushing the tip of the ventricle. This asymmetry in the vortex development seems to be crucial for the function of the ventricle, since without it the elongated shape cannot be flushed efficiently as a symmetrical ring vortex would stay in the wide part of the ventricular cavity leaving a stagnant area in the apex. The understanding of the relation between ventricular shape and motion and the asymmetry of the developing ring vortex is important for the understanding of the ventricular function as a whole. At the beginning of the systole the vortical structures are only partly dissipated and most of the rotational energy pushes the blood towards the opening aortic valve.

The comparison between the results obtained by the prescribed geometry model KaHMo MRT and the fully fluid-structure-coupled model KaHMo FSI shows good agreement between the two models. Slight differences in the flow occur that can be attributed to equally slight differences between the prediction of the myocardial movement by the structural model and the prescribed movement.

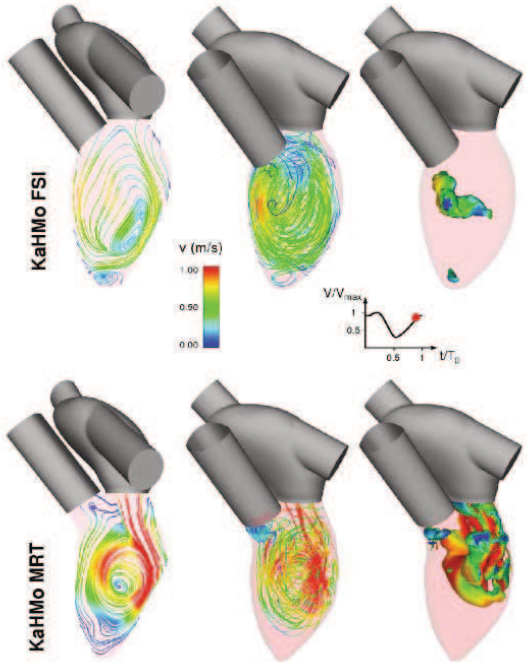

Fig. 2 Comparison between fluid structure interaction and prescribed geometry model [33]

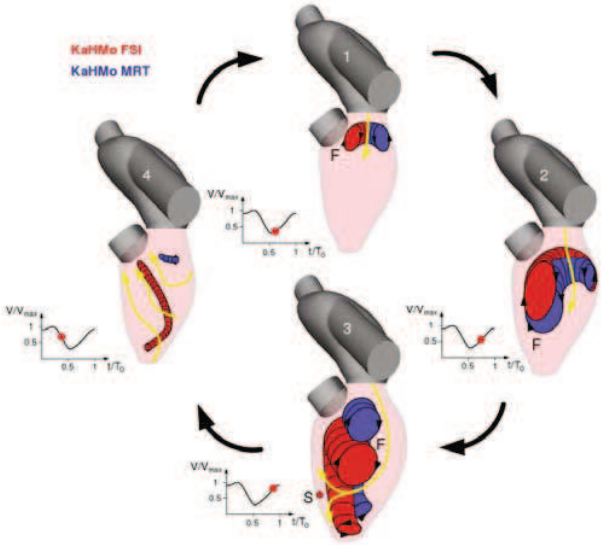

Fig. 3 Schematic intraventricular flow structure for KaHMo FSI (red) and KaHMo MRT (blue) [33]

But it can be said that the general agreement between the two flow fields is good and within a typical uncertainty corridor of 5-10% for this kind of numerical model. Extensive details can be found in [33].

IV. Conclusions

The KaHMo has proven to be suited for simulation of the intraventricular flow field based on patient specific data, that can be obtained by clinical measurements.

The simulation framework is modular and flecible and can be adapted to the demands and necessities. It has been shown that by changing the representation of the myocardium from a prescribed movement to a full fluid-structure-interaction-model the flow model does not need to be changed and gives consistent results between the models.

The flow field that is predicted corresponds to physiological literature and is physically plausible.

The model is extensible. By inclusion of e.g. the electrodynamics of the heart into the FSI model it will be possible to model the change in myocardial movement and thus flow that is caused by an acute myocardial infarction or arrhythmia. The MRT model can be used to evaluate the status quo of healthy and deseased hearts based on direct MRT measurements. This can be used to derive quantities that help to assess the functionality of the heart from a fluid dynamics point of view and to compare different strategies for treatment of e.g. ischemia, ventricular reconstruction, etc.

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REFERENCES

1. Oertel H, Krittian, SBS, Spiegel K (2009) Modelling the human cardiac fluid mechanics. 3rd Edition Universitaetsverlag Karlsruhe in press 2009
2. Schenkel T, Malve M, Reik M et al. (2009) MRI-Based CFD analysis of flow in a human left ventricle: Methodology and application to a healthy heart. Annals of Biomedical Engineering 37(3):503 2009
3. Oertel H (2009) Biofluid mechanics of blood circulation. in Oertel, H (ed.) Prandtl Essentials of Fluid Mechanics. 3rd edition. Springer. New York. 2009
4. Oertel H. (2009) Bioströmungsmechanik. Vieweg-Teubner Verlag Wiesbaden 2009
5. Doenst T, Spiegel K, Reik M et al. (2009) Fluid-dynamic modelling of the left ventricle - prototypical evaluation of the effect of ischemic remodelling and surgical ventricular reconstruction. Annals of Thoracic Surgery. in press 2009
6. Schoephoerster RT, Silva CL, Ray G. (1994) Evaluation of ventricular function based on simulated systolic flow dynamics computed from regional wall motion. J. Biomech. 27:125–136 1994.
7. Taylor TW, Okino H, Yamaguchi T (1994) Three-dimensional analysis of left ventricular ejection using computational dynamics. J. Biomech. Eng. 1994.
8. Saber NR, Gosman AD, Wood NB et al. (2001) Computational flow modeling of the left ventricle based on in vivo mri data: Initial experience. Ann. Biomed. Eng. 29(4):275–283 2001.
9. Saber NR, Wood NB, Gosman AD et al. (2003) Progress towards patient-specific computational flow modeling of the left heart via combination of magnetic resonance imaging with computational fluid dynamics. Ann. Biomed. Eng. 31(1):42–52 2003.
10. Baccani B, Domenichini F, Pedrizzetti G (2002) Vortex dynamics in a model left ventricle during filling. Eur. J. Mech. B/Fluids 21:527–543 2002.
11. Baccani B, Domenichini F, Pedrizzetti G et al. (2002) Fluid dynamics of the left ventricular filling in dilated cardiomyopathy. J. Biomech. 35(5):665–671 2002.
12. Baccani B, Domenichini F, Pedrizzetti G (2003) Model and influence of mitral valve opening during the left ventricular filling. J. Biomech. 36:355–361 2003.
13. Peskin CS, McQueen DM (1996) Fluid dynamics of the heart and its valves, case studies in mathematical modeling, New Jersey: Pretice-Hall, chapter Ecology, Physsiology and Cell Biology: 309–337. 1996.
14. McQueen DM, Peskin CS (2000) A three-dimensional computer model of the human heart for studying cardiac fluid dynamics. Comput. Graph. 34:56–60 2000.
15. Lemmon JD, Yoganathan AP (2000) Three-dimensional comptational model of left heart diastolic function with fluidstructure interaction. J. Biomech. Eng. 122:109–117 2000.
16. Lemon JD, Yoganathan AP (2000) Computational modelling of left herat diastolic function: Examination of ventricular dysfunction. J. Biomech. Eng. 122:297–303 2000.
17. Chahboune B, Crolet JM (1998) Numerical simulation of the blood-wall interaction in the human left ventricle. Eur. Phys. J.-Appl. Phys. 2:291–297 1998.
18. Cheng Y, Oertel H, Schenkel T (2004) Fluid-structure coupled cfd simulation of the left ventricular flow during filling phase. Ann. Biomed. Eng. 33(5):567–576 2004.
19. Vierendeels JA, Riemslagh K, Dick E et al. (1999) Computer simulation of left ventricular filling flow. Comput. Cardiol. 26:177–180 1999.
20. Vierendeels JA, Riemslagh K, Dick E (2000) Computer simulation of intraventricular flow and pressure gradients during diastole. J. Biomech. Eng. 122:667–674 2000.
21. Watanabe H, Hisada T, Sugiura S eta l. (2002) Computer simulation of blood flow, left ventricular wall motion and their interrelationship by fluid-structure interaction finite element method. JSME Int. J. Ser. C- Mech. Syst. Mach. Elem. Manufact. 45(4):1003–1012 2002.
22. Watanabe H, Sugiura S, Kafuku H et al. (2004) Multiphysics simulation of left ventricular filling dynamics using fluid-structure interaction finite element method. Biophysical J. 87:2074–2085 2004.
23. Doyle MG, Vergniaud J-B, Tavoularis S et al. Y (2008) Numerical simulations of blood flow in artificial and natural hearts with fluid-structure interaction, Artificial Organs, 32(11):870-879 2008
24. Perschall M, Spiess H, Schenkel T et al. (2007) Code adapter for arbitrary prescribed motion and deformation using Fluent and MpCCI. Proc. MpCCI 8th User Forum:126-131. Fraunhofer Institute SCAI 2007
25. Nobile, F (2001) Numerical approximation of fluid-structure interaction problems with application to haemodynamics. Dissertation Ecole Polytechnique Federale de Lausanne 2001.
26. Causin P, Gerbeau JF, Nobile F (2005) Added-mass effect in the design of partitioned algorithms for fluid-structure problems. Comp. Meth. Appl. Mech. Engng. 194 (42-44):4506-4527 2005
27. Perktold K, Prosi M, Florian H (2003) Computational models of arterial flow and mass transport. CISM Courses and Lectures. 446:73-136 2003
28. Liepsch D; Thurston G, Lee M (1991) Viscometer studies simulating blood-like fluids and their applications in models af arterial branches. Biorheology 28:39-62 1991
29. Reik M, Meyrowitz G, Schwarz M et al. (2005) 1D circulation model as boundary condition for a 3D simulation of a pumping human heart. IFMBE Proc. vol. 11. World Congress on Med. Phys. & Biomed. Eng. Prague 2005
30. Reik M (2007) Simulation der Strömungsstruktur im menschlichen Herzen. Dissertation Universität Karlsruhe 2007
31. Krittian SBS, Schenkel T, Oertel H (2008) Partitioned FEM/FVM coupling for cardiac fluid-structure interaction with a macroscopic composite approach. Proceedings of The XXII International Congress of Theoretical and Applied Mechanics Adelaide 2008
32. Krittian SBS, Höttges S, Schenkel T et al. (2008) Multi-physical simulation of left-ventricular blood flow based on patient-specific MRI data. Proceedings of The ICBME International Conference on Biomedical Engineering Singapore 2008
33. Krittian SBS (2009) Modellierung der kardialen Strömung-Struktur-Wechselwirkung: Implicit coupling for KaHMo FSI. Dissertation Universität Karlsruhe 2009

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